A CASE OF LEPTO-MENINGITIS CEREBRI PRESENTING TYPICAL SYMPTOMS OF DISSEMINATED SCLEROSIS.[1]

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THE history of this case is as follows: Male, aged —, who gave a history of having had some ten years before specific symptoms consisting of chancre and papula erruption following. Was under my care and treatment at the New York Polyclinic and afterwards in the Hospital for Nervous and Mental Diseases, some three years. During all this time he presented all the symptoms of a case of disseminated sclerosis, and as such he was observed with interest by my former assistants, Dr. B. Sachs and Dr. M. Allen Starr, and I have also repeatedly lectured upon him as an illustration of this disease. To summarize the history, which is very full and minute, as it extends through several years of my case books, it will suffice to say: The tremor was of the voluntary type, this characteristic persisting to the day of death. In sitting still there was none of it. Upon any movement of the muscles, or upon any excitement, it would be observed. This tremor affected both upper and both lower extremities. The tongue also was tremulous. Both the tremor of the tongue and of the extremities was of the jerky kind which is generally seen in well-marked cases of disseminated sclerosis. The muscles of the face were also extremely tremulous when the patient spoke. There was well-marked nystagmus. The speech was of the scanning, jerky variety, which, in the first year of my observation of the patient, did not interfere with articulation further than by rendering it tremulous. The patellar tendon reflexes

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were always somewhat exaggerated. When the patient first came to my clinic, these were all the symptoms. His memory was good, as was also his intelligence. The statement made by himself and his wife was that the disease had been gradually coming upon him for a period which they indefinitely expressed as two or three years. There was no history whatsoever of any mental trouble or, indeed, of any alteration in the patient's mental characteristics. There was no history of cephalalgia or trauma. A careful examination of the different cranial nerves, as well as the sensations of tact, pain, temperature, and muscularity in the trunk, head, and limbs, showed nothing abnormal; there was no neuro-retinitis at any time in the course of the case. The man was taken into the wards of the Hospital for Nervous and Mental Diseases, and kept under constant observation for nearly three years. During this time he had a number of apoplectoid attacks, characterized by a temperature up to 102, 103, once 104 even, lasting for twenty-four or thirty-six hours; violent emesis, usually at the beginning of the attack; great dilatation of the capillaries of the skin of the face; a curious chattering of the lower jaw against the upper, that would come on in paroxysms throughout the attack; occasionally a violent generalized convulsion with loss of consciousness, that was sometimes repeated in the same attack. During these attacks the patient would be so weak that he could not rise from the bed, and this condition of motor weakness would last for several days, passing off gradually. Upon no one of these occasions was there any paralysis induced, motor or sensory. During these attacks the patient's face bore an anxious, distressed look, and, over and above the increased tremor of the tongue and face, there was evidently a motor inability of the patient to articulate. For this reason it was impossible to judge as to the amount of mental confusion, the more especially as the patient maintained, after the attack had passed off, that he was perfectly aware of all that had taken place during it. The convulsive phenomena, as well as the chattering of the lower jaw, were seemingly relieved quickly by the administration of bromides and hypodermics of morphia. In the

course of a year the patient's mind very gradually became more and more impaired by passage into a simple dementia, without hallucinations, illusions, or delusions of any kind. A general motor weakness gradually made itself apparent, but never progressed beyond a slight degree until two or three weeks of death; thus, when being brought across in a car on the Brooklyn Bridge, the slight jar usually felt in the train at the moment of stopping was sufficient to cause the man to fall full length upon the floor. At the same time, within six months of his death, he was strong enough motorially to wander out of the hospital, travel several miles around the city, and so effectually lose himself that it took us several days to find him again. Toward the last he grew very much demented, and so obnoxious did he therefore become in his habits that it required almost the sole attention of one nurse. This extreme condition of dementia existed some four months before his death, and yet his motor weakness had not progressed beyond the degree that I have stated, and did not progress until within three weeks of his death, when he remained most of the time in bed, and on attempting to walk was likely to stumble against any object in the way and fall, although even at this time he moved his limbs so freely and persistently that it was almost impossible to keep any clothing upon him. It is probable that the apparent motor weakness of these latter days was due in greater degree to the dementia than to any great injury to the motor fibres. During the period of dementia it was, of course, impossible to make any accurate examination of the different senses, although we could, of course, perceive in a general way that his sight and hearing were good. Death came about by gradual æsthenia, and was quiet and painless.

That this case was one of disseminated sclerosis, no one can doubt. The characteristic voluntary tremor, the peculiar scanning speech, and the nystagmus were three symptoms persisting for several years that almost settled the diagnosis of themselves. The differential diagnosis was carefully considered from general paresis and intercranial specific trouble. General paresis was excluded because of the

lack of any characteristic mental symptoms or pupillary irregularity, as well as because the speech and the tremor were not those of this disease; nor at any time in the course of the disease were there any symptoms that led us to doubt these conclusions. Intrcranial specific trouble was excluded because of the lack of any cephalalgia or insomnia, or paralysis or affection of any of the cranial nerves. The typical tremor of the purest voluntary type, the scanning speech, the nystagmus, the age of the patient, the upright gait, and the remarkable persistence of these symptoms until the very last, were certainly facts upon which alone a diagnosis of disseminated sclerosis could be made, unless we are to discard entirely our diagnostic criteria of this disease, as they have been made classic by the descriptions of every writer upon the subject. Paralysis agitans, it is hardly necessary to say, was excluded for the reasons that have been given above, as well as because of the lack of the characteristic attitude and the characteristically deliberate speech.

The autopsy was made twelve hours after death. The blood was very fluid. Brain substance was found to be soft and œdematous. There was a great excess of fluid in the ventricles. The dura mater was normal. There was a severe lepto-meningitis over all the vertex and the cerebrum, extending down on either side to the tempora-sphenoidal lobes, so as to implicate irregularly the second temporal convolutions; at the base of the brain the pia mater seemed normal over the bases of the frontal lobes and backward over the bases of the cerebrum and cerebellum.

Careful search was made for patches of sclerosis, but none were found; nor was there at any point the hyperæmia upon contact with the oxygen of the air which has been so often observed in sclerotic patches. Sections were also carefully made of the cerebrum, each section varying in thickness from a half inch to an inch, so as to permit of subsequent microscopical examination; and especial care was observed in examining around the walls of the cerebral ventricles, but at no point was a sclerotic patch found. The pons,

the medulla oblongata, and the spinal cord were stripped of the dura mater, and their external surfaces also examined, but no evidences of sclerosis were found in any of them. Sections throughout the cerebellum were equally negative.

The only case in any way similar to this which I can find in the medical literature, is one detailed by Koenig before the Berliner Gesellschaft für Psychiatrie und Nervenkrankheiten, Sitzung von Jan. 10, 1887, relating to a man thirty-three years old, who fell in his twelfth year upon the left side of his head, was rendered unconscious, and only became able to walk and speak after several months. Later on there appeared dementia and the following phenomena: dragging of the right leg, slight right facial paresis, distinct evidences of motor aphasia, diminution of the perception of pain on the right side, marked tremor of the extremities upon voluntary motion; patella tendon reflex present on both sides; finally complete dementia and death from pneumonia. At the autopsy there was found, together with old hæmatoma of the dura, chronic lepto-meningitis, hydrocephalus, and atrophy of the convolutions from sclerosis that was also recognizable microscopically. This case and mine are certainly very strong evidence in favor of the view that the tremor of disseminated sclerosis is due to lesion of the cortex or of the underlying white strands. Unger in his recent monograph upon multiple sclerosis in children, and Hyman in his late brochure upon paralysis agitans, call attention to numerous facts supporting this theory. The former reminds us that in the diffuse form of cerebral sclerosis in children, Erb and Steffen and other authors have observed tremor either of the voluntary or permanent type. Larcher reported a case of diffuse sclerosis principally affecting the pons, in which there was no tremor. Westphal treated a case of paralysis agitans in which all four extremities were tremulous. In the course of the disease the patient became hemiplegic, and thenceforward the paralytic remained motionless, whilst the tremor continued in the other limbs. As Hyman observes, this must mean that the lesion inducing hemiplegia also interrupted conduction from the cortex. This was confirmed by finding at the autopsy

a hæmorrhage in the internal capsules. Parkinson also mentions a case in which certain limbs becoming paralyzed ceased to be tremulous, but in which the tremor was renewed as the paralysis disappeared. Grashey describes a patient in whom a general tremor ceased upon the supervention of a light hemi-paresis of the right side. In a late discussion in the New York Neurological Society, Dr. Starr mentioned that the rate of vibration in paralysis agitans was eight per second, as had been demonstrated by Gowers, and that cortical irritation of a moderate degree in monkeys produced muscular tremor also characterized by eight vibrations per second.

DISCUSSION.

Dr. WEBBER referred to a case of disseminated sclerosis in which the tremor was entirely wanting, the case having been diagnosed as one of locomotor ataxia. Tremor was supposed to be produced by sclerosis of the medulla or pons, and the explanation was that the parts producing tremor were not affected. The case of Dr. Gray's was interesting as presenting tremor with a lesion of the cortex.

Dr. BANNISTER considered tremor due to interference with conduction, not to the cortical affection *per se*.

Dr. MILLS thought that those cases should make us more careful in the analysis of symptomatology. Even some symptoms regarded as pathognomonic were probably not due so much to the nature of the lesion as to its location in the cerebro-spinal axis. Tremor shows a want of co-ordination. Disseminated sclerosis might exist in the sensory tracts without tremor. Lepto-meningitis was an irritative lesion which interfered probably with the initiation of impulses.

Dr. PUTNAM suggested that the tremor in Dr. Gray's case was similar to that found in old cases of cerebro-spinal meningitis. This was a species of intention tremor.

Dr. GRAY replied that he had in his mind a picture of the tremor of meningitis, and that it did not correspond to that in his case. He had seen also a continuous tremor in disseminated sclerosis and an intention tremor early in par-

alysis agitans. This man had a voluntary tremor throughout and scanning speech. The question of diagnosis lay between intracranial syphilis, disseminated sclerosis, and general paralysis of the insane. In many cases of general paresis, however, there was no implication of the pia. In his opinion, the lesion of general paresis was an interstitial encephalitis, not a meningeo-encephalitis at all. One year ago the speaker had had a case of lepto-meningitis of the whole tempero-sphenoidal lobe. The patient was perfectly sane, loss of memory being the only symptom. In view of these two cases, the speaker inquired what then could be considered the symptoms of lepto-meningitis. The apoplectoid attacks present in the case, the subject of the paper, had been laid down by Charcot as especially characteristic of disseminated sclerosis.